Abdominal Decompression in Pregnancy

Obstetrics is peculiarly open to ideas for treatment which superficially seem slightly eccentric yet catch the imagination of pregnant women. One example which has confused the obstetric scene for the last 15 years is abdominal decompression. In 1959 Heyns of Johannesburg described the use of intermittent abdominal decompression in labour.[1] His interest dated from the observation that the first stage of labour could be facilitated by maternal curarisation. He concluded that this was because the uterus, unconstrained by tension in the abdominal wall, was rising with contractions to lie in line with the pelvic axis and thus was able to act more efficiently.

Later the technique was used during the last 12 weeks of pregnancy, and a large number of claims were made for its effects. Decompression was said to shorten labour and relieve its pain,[1] be effective in treating pre-eclampsia,[2] preventing pre-eclampsia,[3] and preventing and treating fetal asphyxia[4] while producing babies "far superior" mentally and physically.[6] Clearly anything which could achieve all this would be the answer to obstetricians' prayers—and those of their patients. That it has not become established practice in obstetric units throughout the world indicates some general reservations about the evidence upon which these wide-ranging claims were based—mainly related to lack of randomized controls assessed on a "blind" basis. Acceptance by the profession was certainly not helped by the fact that early claims were publicized in women's magazines. An additional factor was probably the cumbersome, constrictive, and noisy nature of the apparatus.

Statistically valid trials certainly presented difficulties—a double blind comparison seemed out of the question in view of the obvious nature of the apparatus and the patients' own awareness of the negative pressure. Women asking for decompression came generally from social classes I and II and so were inherently more likely to produce babies of good quality. Objective but indirect evidence was produced by studies using isotope techniques,[5 6] which showed increased counts over the placental site at the time of decompression. It is, however, a considerable extension to conclude from the evidence of increased counts at the moment of decompression applied for a ten thousandth part of the total time in late pregnancy that a sustained effect is produced throughout the period. It was also claimed[7] that the total excretion of pregnanediol and oestriol—accepted indicators of fetoplacental "well-being"—was increased by abdominal decompression.

Recently, however, a study has been reported[8] in which an ingenious approach was made to the problem of controls. All patients were given decompression but in one group the pressure was only −20 mm Hg while in the other it was −70 mm Hg, the order of pressure used in previous work. It was assumed that the lower level would certainly be ineffective and therefore acceptable for control purposes. The study included 411 primigravidae, 200 treated at −70 mm Hg and 211 at −20 mm Hg. At the end of the study the mean placental weight was significantly higher in the controls—contrary to what might have been expected if decompression exerted a beneficial effect; mean birth weight was also higher in the control group, but not significantly so. There was only one stillbirth in the control group, associated with a short cord encircling the baby's neck, but three in the high decompression group, one being due to anencephaly but two being unexplained. The admission rate for pre-eclampsia was almost identical in the two groups, though the high decompression group had a significantly shorter hospital stay when admitted for pre-eclampsia. The admission rate for pre-eclampsia was lower than expected in both groups, suggesting that either the low decompression regimen had an influence or that patients at low risk of developing pre-eclampsia selected themselves for the trial. The authors inclined to the latter view, and this is in keeping with the interpretation placed by many obstetricians on earlier data. The conclusion reached was that decompression did not apparently produce a reduction in blood pressure in pregnancy or significantly influence the characteristics of pregnancy and labour or the size and maturity of babies.

The plan of this study and its essentially negative findings would be likely to cause most obstetricians to regard the abdominal decompression story as at an end and consigned to obstetrics' large historical collection of oddities. However, in the same issue of the same journal there was another report on a smaller number of cases with somewhat contrary conclusions.[9]

This was a study of 140 pregancies in which the fetus seemed "small-for-dates"—by nature an extremely ill-defined group, but clinical grading was supported by ultrasonic cephalometry. The trial was not a blind one, and despite initial randomization seven patients allocated to the decompression group were transferred at their own request to the control group and this may have had an influence. Decompression was given daily for 30 minutes using −80 to −90 mm Hg for 25 seconds in each minute. There was a higher incidence of labour induction for placental insufficiency in the control group (27 as opposed to 20), though there was no significant difference in maturity at delivery. There was a higher incidence of forceps deliveries for fetal distress in the control group. The main feature on which a significant effect of the decompression regimen was claimed, however, was a relatively greater growth rate of the biparietal diameter as measured ultrasonically: 2·08 mm per week as opposed to 1·49 per week ($P < 0·001$). There was also a greater increase in urinary oestriol, and the babies in the treated group were significantly heavier ($P < 0·001$). It was concluded that an unequivocal beneficial effect was exerted on the small-for-dates fetus by the decompression regimen.

It could be said that the two studies with their application of scientific methodology to the problem leave the question just about as confused as before. That of Coxon *et al.*,[8] however, was more subtle, larger, and double-blind with randomization. Supporters of decompression will no doubt attribute the lack of demonstrable influence to the very complexity of the trial protocol, but most obstetricians are likely to be more sceptical. Unfortunately some will still find it impossible to say categorically that it is valueless to patients who come inquiring about it or demanding it.

How might decompression work if it is effective? It was initially suggested that it improved blood flow directly through the intervillous space—just as peripheral limb blood flow may be improved by negative pressure. An alternative possibility would be that by allowing the uterus to come further forward, away from the inferior vena cava, the negative pressure may indirectly allow improved drainage from the intervillous space and therefore improve circulation. As this can be achieved by simple postural means[10] it would only be reasonable and rational to take full advantage of this easy approach before resorting to the inhibiting complexities of the decompression regimen.

If eventually it does seem that decompression in pregnancy exerts a beneficial effect difficult clinical questions will arise,

to which the answers would be largely philosophical. If it is effective when applied to some for short spells it could presumably be much more effective if applied to all for long periods or continuously. That would put decompression in a similar category to the possibility of enhancing cerebral acuity and diminishing risk of infection by living continuously in a sterile chamber of hyperbaric oxygen—scientifically possible but on a commonsense basis nonsensical. For most obstetricians and their patients nature's abhorrence of a vacuum seems likely to prevail.

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Methoxyflurane Nephrotoxicity

Whenever a new anaesthetic agent is introduced there is always a period when it is hailed as the answer to every anaesthetist's prayer. Methoxyflurane is hardly an exception, though it started with the great disadvantage that its blood-to-gas solubility ratio is as high as 13·0, which means that the necessary tension in the brain to produce anaesthesia builds up slowly and an equally long time is needed for the patient to recover consciousness. In this respect methoxyflurane resembles trichloroethylene; but since it also has a very high lipoid solubility relatively low concentrations can be used to maintain surgical anaesthesia. Furthermore, these low concentrations do not irritate the respiratory tract, so that methoxyflurane has proved to be a useful anaesthetic agent in clinical practice.

Soon after its clinical debut there was a report[1] that methoxyflurane might produce renal damage. In 1971 Mazze and his colleagues,[2] in a controlled study, provided evidence that when the drug was given alone it could cause polyuric renal insufficiency. No precise dose-effect relationship was established, so it was not possible to say whether methoxyflurane when used in low concentrations in combination with other anaesthetic agents was likely to produce renal damage or not. Now Cousins and Mazze have taken the matter a stage further and have conclusively shown[3] that when the concentration exceeds a certain level (2·0 M.A.C. hours—minimum anaesthetic concentration for surgical anaesthesia multiplied by the duration of the anaesthesia) then some renal damage occurs. Animal studies have also shown that if methoxyflurane is used in the presence of other potentially nephrotoxic drugs such as gentamicin[4] or the tetracyclines[5] the effect is additive.

Methoxyflurane produces two important metabolic products in vivo—inorganic fluoride and oxalic acid. Fluoride is known to inhibit several enzyme systems and chronic ingestion leads to renal damage. It has been suggested that the renal damage is produced principally by fluoride inhibition of the enzyme systems necessary for sodium pumping in the ascending loop of Henle or early distal tubule.[6] Oxalic acid has been ruled out as the causative agent since the type of renal damage and also the clinical signs in these cases were different from those of oxalic acid intoxication.

The result of many careful studies has been to establish without any doubt that if patients breathe a high concentration of methoxyflurane for an hour or more there is an increasing risk of renal damage, even in the presence of normal function. Fortunately, methoxyflurane has never found wide acceptance as an anaesthetic agent in the United Kingdom. It has, however, been shown to be useful when given in low concentrations for relief of pain in midwifery.[7-9] Though no harm has yet been shown to result from the use of these low concentrations in the presence of normal renal function, it does seem clear that if the use of methoxyflurane is essential a careful watch must be kept on both the concentration and the duration of administration.

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Gonorrhoea of the Pharynx

In April 1970 Bro-Jørgensen and Jensen cultured gonococci from the pharynx of a man who was symptom-free a few days after an orogenital contact. This led them to culture pharyngeal material routinely for gonococci from all their patients in Copenhagen with suspected venereal disease. They have now[1] completed tests on 804 men and 542 women infected with gonorrhoea and similar numbers free from that disease, a total of some 2,700 persons.

Culture of the pharynx showed *Neisseria gonorrhoeae* in 110 of these patients: 55 women, 43 heterosexual men, and 12 homosexual men. The pharynx was the only site affected in eight of the women, six of the heterosexual men, and two of the homosexual men. Among patients suffering from gonorrhoea, pharyngeal infection was found in 10% of the women, 7% of the heterosexual Danish men, 2% of the heterosexual foreign men, and no fewer than 25% of the homosexual men.

A consecutive series of 1,203 heterosexual Danish patients (523 women, 680 men) were asked about orogenital contact. The pattern of behaviour was similar in men and in women and in those with and without gonorrhoea. About 20% claimed never to have had orogenital contact, 75% had it frequently or fairly often, and about 35% at the most recent sexual contact. Orogenital contact was almost the rule for homosexual men; this was reflected in the high rate of pharyngeal infection in members of this group with gonorrhoea.

Pharyngeal gonorrhoea was usually clinically silent: 79% of patients were symptom-free. The others had sore throat with tonsillitis of varying degree, occasionally with exudates